Complete Summary

GUIDELINE TITLE

Allergy diagnostic testing: an updated practice parameter. Part 1.

BIBLIOGRAPHIC SOURCE(S)

Bernstein IL, Li JT, Bernstein DI, Hamilton R, Spector SL, Tan R, Sicherer S, Golden DB, Khan DA, Nicklas RA, Portnoy JM, Blessing-Moore J, Cox L, Lang DM, Oppenheimer J, Randolph CC, Schuller DE, Tilles SA, Wallace DV, Levetin E, Weber R, American Academy of Allergy, Asthma and Immunology, American College of Allergy, Asthma and Immunology. Allergy diagnostic testing: an updated practice parameter. Part 1. Ann Allergy Asthma Immunol 2008 Mar;100(3 Suppl 3):S15-S66.

GUIDELINE STATUS

This is the current release of the guideline.

This guideline updates a previous version: Joint Council of Allergy, Asthma and Immunology. Practice parameters for allergy diagnostic testing. Ann Allergy Asthma Immunol 1995 Dec;75(6 Pt 2):543-625. [316 references]

COMPLETE SUMMARY CONTENT

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SCOPE

DISEASE/CONDITION(S)

Human hypersensitivity disorders (allergies):

- Immediate hypersensitivity reactions
- Delayed hypersensitivity reactions

- Cell-mediated immune conditions

GUIDELINE CATEGORY

Diagnosis
Evaluation
Screening

CLINICAL SPECIALTY

Allergy and Immunology

INTENDED USERS

Physicians

GUIDELINE OBJECTIVE(S)

- To serve as a reference source for current utility and validity of allergy diagnostic tests
- To develop a reliable reference resource for selecting appropriate diagnostic tests
- To provide guidelines and support for the practicing physician on how diagnostic tests should be used in an appropriate and cost-effective manner
- To improve the quality of care of patients by facilitating prompt and accurate diagnosis of their hypersensitivity disorders

TARGET POPULATION

Children and adults with hypersensitivity disorders (allergies)

INTERVENTIONS AND PRACTICES CONSIDERED

1. In vivo diagnostic tests of immediate hypersensitivity reactions
    - Percutaneous in vivo diagnostic skin tests
    - Intracutaneous in vivo diagnostic skin tests
    - Reading and interpreting late-phase cutaneous responses
2. Organ challenge tests
    - Conjunctival challenge test
    - Nasal challenge test
    - Specific bronchial challenge test
    - Occupational challenge test and evaluation at and away from work
    - Evaluation of inflammatory biomarkers of upper and lower airway fluids
3. Tests to distinguish clinical obstructive diseases resembling asthma: cystic fibrosis and alpha^{1}-trypsin deficiency
4. In vivo diagnostic tests of cell-mediated immunity
    - Tuberculin and recall intracutaneous tests
    - Epicutaneous patch test
    - Modified epicutaneous atopy patch test and repeated use test
5. In vitro diagnostic tests of immediate hypersensitivity

- Total serum immunoglobulin E (IgE) assays
- Allergen specific IgE assays
- Allergen specific immunoglobulin G (IgG) and IgG subclass assays
- Histamine and leukotriene tests
- Measurement of plasma tryptase levels
- Measurement of eosinophils, eosinophil-derived substances and chemoattractants in body fluids
- Basophil activation test

6. In vitro diagnostic tests of cell-mediated immunity
7. Other diagnostic immunologic tests
8. Unproven tests

MAJOR OUTCOMES CONSIDERED

- Clinical utility and validity of diagnostic tests (i.e., sensitivity, specificity, and positive and negative predictive values)
- Limitations of diagnostic tests
- Safety of tests

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

The draft was based on a review of the medical literature using a variety of search engines, such as PubMed.

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Expert Consensus
Weighting According to a Rating Scheme (Scheme Given)

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Category of Evidence

Ia Evidence from meta-analysis of randomized controlled trials

Ib Evidence from at least 1 randomized controlled trial

IIa Evidence from at least 1 controlled study without randomization

IIb Evidence from at least 1 other type of quasi-experimental study

III Evidence from nonexperimental descriptive studies, such as comparative studies, correlation studies, and case-control studies

IV Evidence from expert committee reports, the opinion or clinical experience of respected authorities, or both

LB Evidence from laboratory-based studies

METHODS USED TO ANALYZE THE EVIDENCE

Systematic Review

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Published clinical and basic studies were rated by categories of evidence and used to establish the strength of recommendations (see "Rating Scheme for the Strength of the Evidence" and "Rating Scheme for the Strength of the Recommendations" fields).

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Expert Consensus

DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS

The major emphasis of this updated version of Practice Parameters for Allergy Diagnostic Testing is focused on how technological refinements and their validations during the past decade are being incorporated into the diagnostic armamentarium of allergists/clinical immunologists and how their optimal use enables confirmation of human clinical sensitivity.

The impetus for Practice Parameters for Allergy Diagnostic Testing originally stemmed from a consensus conference sponsored by the National Institute of Allergy and Infectious Diseases and published as a supplement to the *Journal of Allergy and Clinical Immunology* in September 1988. One of the major conclusions of that workshop was that periodic reassessment of diagnostic techniques should be mandatory, and in keeping with that recommendation, the 1995 Practice Parameters for Allergy Diagnostic Tests further reviewed and considered new developments up to that time. In the 13-year interval since that publication, there has been an exponential progression of basic and translational immunologic research, some of which produced novel and practical diagnostic possibilities. Obviously, these advancements necessitated an overhaul of the 1995 Allergy Diagnostic Parameter commensurate with the extensive database currently available. The ultimate goals were to formulate recommendations based on evidence-based literature and to achieve balanced use of classic and new diagnostic methods.

The working draft of the Parameter on Allergy Diagnostic Tests update was based on an outline jointly conceived by the co-chairmen of the Parameter Workgroup and realized by the work group.

Many of the diagnostic recommendations were extracted or in some cases quoted verbatim from each of these previously published guidelines. Disease Management of Drug Hypersensitivity: A Practice Parameter; Allergen Immunotherapy: A Practice Parameter; Stinging Insect Hypersensitivity: A Practice Parameter; Food Allergy: A Practice Parameter; and Contact Dermatitis: A Practice Parameter.

This document represents an evidence-based, broadly accepted consensus opinion.

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Strength of Recommendations

A Directly based on category I evidence

B Directly based on category II evidence or extrapolated from category I evidence

C Directly based on category III evidence or extrapolated from category I or II evidence

D Directly based on category IV evidence or extrapolated from category I, II, or III evidence

E Directly based on category LB evidence

F Based on consensus of the Joint Task Force on Practice Parameters

NR Not rated

COST ANALYSIS

Published cost analyses were reviewed in the preparation of this guideline.

METHOD OF GUIDELINE VALIDATION

External Peer Review
Internal Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

The initial draft was reviewed by all members of the Joint Task Force and subsequently by the American Academy of Allergy, Asthma and Immunology (AAAAI), the American College of Allergy, Asthma and Immunology (ACAAI), and the Joint Council of Allergy, Asthma and Immunology and a number of experts on

in vivo and in vitro diagnostic immunology selected by the supporting organizations. Comments were also solicited from the general membership of these societies via their Web sites. The peer review process and general format of the Practice Parameter are consistent with recommendations of the American College of Medical Quality, which defines practice guidelines.

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

Guideline recommendations are presented in the form of summary statements. After each statement is a letter in parentheses that indicates the strength of the recommendation. Grades of recommendations (A-D) and categories of evidence (Ia, Ib, IIa, IIb, III, IV, LB [evidence from laboratory-based studies], and NR [Not rated]) are defined at the end of the "Major Recommendations" field.

Summary Statements

In Vivo Diagnostic Tests of Immediate Hypersensitivity Reactions

Percutaneous and Intracutaneous In Vivo Diagnostic Skin Tests

1. First described in 1867 by Dr Charles Blackley, skin tests (prick/puncture and intracutaneous) have evolved as reliable, cost-effective techniques for the diagnosis of immunoglobulin E (IgE)-mediated diseases. (**B**)

Prick/Puncture Tests

2. Prick/puncture tests are used to confirm clinical sensitivity induced by aeroallergens, foods, some drugs, and a few chemicals. (**B**)

Technique

3. A number of sharp instruments (hypodermic needle, solid bore needle, lancet with or without bifurcated tip, and multiple-head devices) may be used for prick/puncture tests. (**C**)
4. Although a number of individual prick/puncture comparative studies have championed a particular instrument, an objective comparison has not shown a clear-cut advantage for any single or multitest device. Furthermore, interdevice wheal size variability at both positive and negative sites is highly significant. (**C**)
5. Optimal results can be expected by choosing a single prick/puncture device and properly training skin technicians in its use. (**C**)
6. Although prick/puncture tests are generally age, sex, and race independent, certain age (children younger than 2 years and adults older than 65 years) and racial (African American children) factors may affect their interpretation. (**C**)
7. Skin test allergens used for prick/puncture tests should be potent and stable. (**B**)

8. To ensure proper interpretation, positive (histamine) and negative (saline or 50% glycerinated human serum albumin [HSA]–saline) should be performed at the same time as allergen tests. (**B**)

Reading the Test Results

9. The peak reactivity of prick/puncture tests is 15 to 20 minutes at which time both wheal and erythema diameters (or areas) should be recorded in millimeters and compared with positive and negative controls. (**B**)
10. Qualitative scoring (0 to 4+; positive or negative) is no longer used by many clinicians because of interphysician variability in this method of scoring and interpretation. (**B**)

Clinical Relevance

11. The diagnostic validity of prick/puncture tests has been confirmed not only in patients exposed to allergens under natural conditions but also in patients undergoing controlled organ challenge tests. (**B**)
12. Although prick/puncture testing often correlates with exposure history, there are significant exceptions to this observation. (**B**)

Sensitivity, Specificity, and Positive and Negative Predictive Indices

13. Many studies have verified the sensitivity and specificity of prick/puncture tests for both inhalant and food allergens when correlated with nasal and oral challenge tests. (**B**)
14. Compared with clinical history alone, the diagnostic accuracy of prick/puncture tests showed more limited capacity to predict clinical sensitivity for both inhalant and food allergens. (**C**)

Limitations

15. The reliability of prick/puncture tests depends on the skill of the tester, the test instrument, color of the skin, skin reactivity on the day of the test, potency, and stability of test reagents. (**C**)
16. False-positive prick/puncture tests may occur (1) to tree pollens in honey bee–sensitive patients due to cross-reactive carbohydrate determinants present in honey bee venom and (2) in tree-sensitive patients being tested to tree pollens no longer indigenous to the area. (**C**)
17. The rare occurrence of specific positive organ challenge test results in patients with both negative prick/puncture and intracutaneous tests suggests that alternative pathways, including locally secreted IgE, IgE-independent, or nonimmune stimuli may activate mediator release in the end organ. (**C**)

Safety

18. Life-threatening generalized systemic reactions are rarely caused by prick/puncture tests. In a recent retrospective survey, 1 death was reported in a patient who received 90 food prick/puncture tests at one time. (**C**)

Intracutaneous Tests

Present Applications

19. Intracutaneous tests will identify a larger number of patients with lower skin test sensitivity and are used when increased sensitivity is the main goal of testing. (**B**)
20. Intracutaneous tests are useful for evaluation of anaphylaxis, particularly drug (i.e., penicillin) and *Hymenoptera* venom anaphylaxis. (**A**)
21. When compared with specific nasal challenge, skin end point titration (SET) is equivalent to prick/puncture skin tests. (**B**)

Techniques

22. Intracutaneous tests should be performed with small volumes (approximately 0.02 to 0.05 mL) of allergens injected intracutaneously with a disposable 0.5- or 1.0-mL syringe. (**C**)
23. As a general rule, the starting dose of an intracutaneous allergen test ranges from 100- to 1,000- fold more dilute than the allergen concentration used for prick/puncture tests. (**C**)

Reading the Test Results

24. Intracutaneous tests are read 10 to 15 minutes after injection, and both wheal and erythema (in millimeters) should be recorded. (**B**)

Clinical Relevance

25. The diagnostic sensitivity of intracutaneous tests is probably greater than prick/puncture tests when testing for penicillin, insect venom, or certain drug class (e.g., insulin, heparin, muscle relaxants) hypersensitivity. (**C**)
26. The greater sensitivity of titrated intracutaneous tests, especially in the erythema component, is an advantage for determining biologic potency of allergen extracts and biologic allergy units (BAU) as based on intracutaneous erythema assays in sensitive human volunteers. (**B**)

Sensitivity, Specificity, and Positive and Negative Predictive Indices

27. At dilutions between 10^{-2} and 10^{-3} (weight/volume [wt/vol]), intracutaneous tests for most allergens exhibit poor efficiency in predicting organ challenge responses and correlating with the presence of detectable serum specific IgE. (**C**)
28. There are limited data about equivalency of sensitivity, specificity, and predictive indices between intracutaneous and prick/puncture tests when compared with organ challenge tests. One study demonstrated that more dilute intracutaneous concentrations were comparable to prick/puncture tests in predicting positive nasal challenges. (**C**)
29. Similar comparative equivalency studies based on history and symptoms alone revealed that intracutaneous tests were comparable to prick/puncture tests only at intracutaneous titration end points between 10^{-5} and 10^{-6} g/mL (wt/vol). (**B**)
30. Because clinical use of intracutaneous tests is usually restricted to a single dose (i.e., 1:1,000 wt/vol), which may be irritant, predictive accuracy of

these tests at this concentration is often confounded by false-positive results. (**C**)

Limitations

31. For most allergens, a fixed dilution (1:1,000 [wt/vol]) of intracutaneous tests has poor efficiency in predicting organ challenge responses. (**A)**
32. Intracutaneous tests are occasionally negative in venom-sensitive patients who experience life-threatening reactions. (**C**)
33. Repetitive (≥2) intracutaneous penicillin testing may sensitize a small number of individuals to penicillin. (**C**)

Safety

34. Immediate systemic reactions are more common with intracutaneous tests; 6 fatalities were reported in a recent retrospective survey. (**C**)
35. Prescreening with prick/puncture tests is a practical way to avoid life-threatening reactions to intracutaneous tests. (**C**)
36. If prick/puncture prescreening is not used, preliminary serial threshold titrations should be considered, starting at high dilutions (10^{-5} to 10^{-8} g/mL [wt/vol]). This is of particular importance if exquisite sensitivity (e.g., anaphylaxis to foods and drugs) is suspected. (**D**)

Late-Phase Cutaneous Reactions

Definition and Description

37. The late-phase cutaneous response is a continuation of either prick/puncture or intracutaneous testing, generally the latter, and is characterized by erythema, induration or edema, and dysesthesia. (**B**)

Causes

38. The late-phase cutaneous response may occur after both immune and nonimmune activation. Many allergens have been implicated. (**B**)

Reading Tests Results

39. The late-phase cutaneous response should be read between the 6th and 12th hours after the skin tests are applied; measurements of mean diameter and/or area of induration or edema should be recorded. (**B**)

Clinical Relevance

40. Although the clinical relevance of late-phase cutaneous response is not as yet fully established, several randomized, controlled studies suggest that reduction in sizes of late-phase cutaneous response may parallel clinical response to immunotherapy. (**B**)

Safety

41. The same principles that pertain to safety of skin tests apply to late-phase cutaneous responses. (**C**)

Inhibitors of the Late-Phase Cutaneous Response

42. Preadministration of drugs, such as calcineurin inhibitors, misoprostol, prednisone, and azelastine, before application of skin tests partially or completely inhibit the late-phase cutaneous response. (**B**)

Number of Skin Tests

43. The number of skin tests and the allergens selected for skin testing should be determined based on the patient's age, history, environment and living conditions (e.g., region of the country), occupation, and activities. Routine use of large numbers of skin tests or routine annual tests without a definite clinical indication is clearly not justified. (**D**)

Organ Challenge Tests

Introduction

44. Respiratory challenge tests are used when an objective gold standard for establishing clinical sensitivity is indicated. (**B**)

Conjunctival Challenge

45. Conjunctival challenge tests are usually conducted for suspected localized eye allergy but in some cases they may also be helpful in investigating nasal allergy. (**B**)
46. Conjunctival challenge tests are evaluated by symptoms of itching and objective indices, including tear volume, amount of mucus, and palpebral or bulbar erythema. (**B**)

Nasal Challenge

47. Nasal challenges provide objective evidence of clinical sensitivity when the diagnosis is in question or in situations when it is desirable to evaluate efficacy of therapeutic management. (**B**)
48. Nasal challenge responses are evaluated by subjective symptoms and objective measurements of nasal airway resistance, the number of sneezes, and the measurement of inflammatory mediators in nasal secretions. (**B**)

Specific Bronchial Challenge

49. Specific (allergic) bronchial challenge provides a measure of lower airway clinical sensitivity when there is uncertainty or dispute. (**B**)
50. Guidelines for the performance of specific bronchial challenge include factors such as withholding certain medications before the test, determining the initial allergen dose by preliminary skin or methacholine challenge testing, a beginning forced expiratory volume in 1 second (FEV_1) baseline of 70% or better, the amount or duration of exposure to allergen, measurement of FEV_1

at intervals after the exposure, careful observation for late-phase responses, comparison to a placebo-controlled challenge usually performed the day before the specific challenge, and, optionally, repetition of methacholine challenge 24 to 48 hours after specific challenge for evaluation of induced bronchial hyperresponsiveness. (**B**)

Occupational Challenge Testing

51. Occupational challenge testing requires special precautions with respect to the innate toxicity of the suspected allergen and special apparatuses used to measure and control the quantity of challenge substances, such as potentially irritating volatile agents and dust. (**B**)

Evaluation At and Away from Work

52. A practical clinical method of assessing occupational asthma (OA) is prospective monitoring of the worker at and away from work by serial peak expiratory flow rates (PEFRs) or FEV_1 values if this can be arranged by mutual agreement of employee and employer. (**B**)

Inflammatory Biomarkers of Upper and Lower Airway Fluids

53. Many inflammatory correlates can be evaluated and studied serially in respiratory and other body fluids, such as nasal smears or lavage, induced sputum, and bronchoalveolar lavage (BAL). These may define specific phenotypes or in some cases predict severity. (**B**)
54. Exhaled nitric oxide is a noninvasive measure of airway inflammation and is useful for monitoring objective responses to topically administered corticosteroids. (**B**)
55. Although breath condensate analysis is an evolving noninvasive method for evaluation of asthma, results are still variable and further refinements are required before it can be accepted as a valid diagnostic method. (**C**)
56. Bronchoalveolar lavage obtained through flexible bronchoscopy is useful in phenotyping asthma. The finding of lymphocytic alveolitis may suggest a diagnosis of hypersensitivity pneumonitis. (**B**)

Tests to Distinguish Clinical Obstructive Diseases Resembling Asthma

Cystic Fibrosis

57. Cystic fibrosis may not only be confused with asthma, but certain genetic variants may be associated with increased asthma risks. (**B**)

$Alpha_1$-Trypsin Deficiency

58. Although major phenotypes of $alpha_1$-antitrypsin deficiency do not occur in asthma, recent surveys demonstrated a high prevalence of asthma in young ZZ homozygous $alpha_1$-antitrypsin deficiency patients. (**B**)

In Vivo Diagnostic Tests of Cell-Mediated Immunity

Intracutaneous Tests

Tuberculin and Recall Intracutaneous Tests

59. Purified protein derivative (PPD) of tuberculin is the prototype antigen recall test and provides direct evidence that hypersensitivity, as opposed to toxicity, is elicited by the antigens in *Mycobacterium hominis* or related mycobacterial species. (**B**)
60. The tuberculin skin test is elicited by the intracutaneous injection of 0.1 mL of standardized PPD starting with the intermediate strength of 5 tuberculin units. (**C**)
61. Recall antigen skin tests are used to evaluate cellular immunity in patients with infection (e.g., life-threatening sepsis), cancer, pretransplantation screening, end-stage debilitating diseases, and the effect of aging. (**C**)
62. Reduced or absent recall antigen tests are termed *anergy,* which develops frequently in certain diseases, such as hematogenous tuberculosis, sarcoidosis, and atopic dermatitis. (**C**)

Technique

63. *Candida albicans*, *Trichophyton mentagrophytes*, and *Tetanus* toxoid, the currently available recall antigens, are injected intracutaneously in the same way as the PPD test. (**C**)

Reading the Tests Results

64. The size of the delayed skin test reaction is measured 48 hours after antigen challenge, and the largest diameter of the palpable firm area that outlines the induration response should be measured to the nearest millimeter. (**C**)
65. When a single intracutaneous antigen (other than PPD) is used to evaluate prior sensitization to a potential pathogen, a reaction of 5 mm or greater may suffice as the cutoff point for positive tests, but smaller reactions (2 to 4 mm) may be clinically important. (**C**)

Clinical Relevance

66. The absence of delayed-type hypersensitivity to all the test antigens would suggest an anergic state. (**C**)
67. The most important use of delayed-type hypersensitivity skin testing is epidemiologic screening of susceptible populations exposed to bacterial and fungal pathogens. (**C**)
68. The widest application of recall antigen testing is the detection of anergy and as an in vivo clinical correlate of cell-mediated immunoincompetency. (**C**)
69. Although anergy testing was formerly conducted frequently in human immunodeficiency virus (HIV) patients to determine whether a concurrent negative tuberculin skin test result rules out active tuberculosis, recent evidence mitigates against this approach. Recall antigen anergy in HIV patients has also been investigated as an indicator of staging, progression of disease, and response to therapy. (**C**)

Sensitivity, Specificity, and Positive and Negative Predictive Indices

70. Although the standardized purified protein derivative (PPD) antigen has been used for many years as a predictor of active or latent tuberculosis infection, confounders, such as susceptible populations, Bacillus Calmette-Guérin (BCG) vaccination, and cross-sensitization with other atypical mycobacterial species, have all affected the diagnostic accuracy of the tuberculin skin test and, by extrapolation, other delayed-type hypersensitivity tests. (**C**)

Limitations

71. The gross appearance of a late-phase cutaneous response and delayed-type hypersensitivity reactions may not be completely distinguishable except that the latter are more characterized by prolonged induration. (**B**)
72. Although systemic corticosteroids will render delayed-type hypersensitivity skin tests uninterpretable, 28 days of treatment with high-dose inhaled fluticasone (220 micrograms, 2 puffs twice a day) did not suppress delayed-type hypersensitivity to PPD in healthy volunteers. (**B**)
73. Neither anergy nor tuberculin testing obviates the need for microbiologic evaluation when there is a suspicion of active tuberculosis or fungal infections. (**F**)
74. Several new in vitro assays (i.e., interferon-gamma and polymerase chain reaction) appear to be more reliable in predicting active tuberculosis in BCG-vaccinated persons or when cross-sensitivity to atypical mycobacteria may coexist. (**C**)

Safety

75. Immediate hypersensitivity reactions, including anaphylaxis, have been reported after tuberculin skin tests. (**D**)

Number of Cell-Mediated Hypersensitivity Skin Tests

76. The number of skin tests for delayed, cell-mediated hypersensitivity reactions is relatively limited. (**C**)

Epicutaneous Tests

77. First introduced by Jadassohn in 1896, the epicutaneous patch test has evolved as the definitive diagnostic technique for the diagnosis of allergic contact dermatitis (ACD). (**A**)

Patch Tests

Present Applications

78. When clinical evaluations suggest that exposure to a specific contactant has occurred either in an occupational or nonoccupational clinical setting, patch testing can be used to confirm the diagnosis. (**C**)
79. From a public health perspective, patch testing is useful to identify potential health hazards of unknown and newly introduced contact allergens for the medical community and industrial hygienists. (**C**)

Technique

80. The most common patch test techniques are the individual Finn Chamber and the T.R.U.E. (thin layer rapid use epicutaneous) TEST, a U.S. Food and Drug Administration (FDA)-approved screening method for screening contactant allergens. The T.R.U.E. TEST is preloaded with 23 common contactants and vehicle control that have been previously incorporated into a dried-in-gel delivery system, which is coated onto a polyester backing to form a patch template. (**B**)
81. If photo contact sensitivity is suspected, the appropriate allergens should be subjected to photopatch tests primarily in the ultraviolet-A (UV-A) range of 320 to 400 nm. (**C**)

Reading the Test Results

82. Traditionally, patch tests remain in place for 48 hours. After the 48-hour patch test reading, additional readings at 3 to 4 days and, in some cases, 7 days after the original application of the patch yield the best overall reading reliability. (**C**)
83. A descriptive reading scale developed by 2 major international ACD research groups is the current standard for interpreting patch test results. (**C**)

Clinical Relevance

84. Although patch tests are indicated in any patient with a chronic eczematous dermatitis if ACD is suspected, patch tests are especially important in identifying both irritant contact dermatitis (ICD) and ACD in the occupational setting. (**C**)
85. Other important exposures associated with ACD include the use of topical medication, including corticosteroids, plant-induced ACD, and dermatitis occurring after use of cosmetics and personal hygiene products. (**C**)
86. Unprotected work and repetitive exposure to surfactants may predispose patients to occupational dermatitis, including ICD and ACD. (**C**)
87. Certain contactant allergens in the T.R.U.E. TEST panel, such as nickel and some rubber chemicals, have a high degree of relevant (approximately 75%) correlation with clinical sensitivity but others do not (e.g., hydroxycitronellal, thimerosal). (**B**)

Sensitivity, Specificity, and Positive and Negative Predictive Indices

88. Patch tests are most effective when the patients are selected on the basis of a clear-cut clinical suspicion of contact allergy, and they are tested with the chemicals relevant to the problem; these conditions satisfy the prerequisites of high pretest probability. (**C**)
89. Although the diagnostic accuracy of contactants cannot be compared with other in vivo or in vitro tests, diagnostic concordance between patch test sensitivity and the outcomes of repeated open provocation tests has been demonstrated for some contactants. (**B**)

Limitations

90. The chief limitation to traditional patch testing for the diagnosis of ACD is the lack of a suitable gold standard by which it can be evaluated in terms of diagnostic accuracy predictors and likelihood ratios. (**C**)
91. Other technical limitations of patch tests include the inclusion of relevant contact allergens, use of the proper vehicle, application to the proper skin area, proper reading and interpretation, and the ability to correlate the tests with the patient's specific exposure. (**B**)
92. Other limiting factors concern reproducibility, lack of information about irritant thresholds, and minimal elicitation concentrations (MECs) for many common chemicals in the human environment. (**C**)
93. The inability to separate irritants from allergic responses is often encountered in the angry back syndrome, which occurs in approximately 6% of cases and is likely to develop in patients with a longer duration of the primary dermatitis. (**C**)
94. Negative patch test reactions may occur even when the tests are performed with the correct sensitizing materials because the test fails to duplicate the conditions under which the dermatitis developed (e.g., abrasions, frequent use of irritating soaps, washing the hands with solvents). (**C**)

Safety

95. Systemic ACD after patch testing is rare, as is reactivation of patch test reactions after oral ingestion of related allergens or even by inhalation of budesonide in patients with sensitization to topical corticosteroids. (**B**)
96. It is possible to sensitize a patient who had not been previously sensitized to the allergen being tested. This is particularly true of plant contactants, such as poison ivy or oak and aniline dyes. (**B**)

Modified Epicutaneous Atopy Patch Test (APT) and Repeated Use Test (RUT)

97. Two major variants of traditional patch tests are available: the APT and RUT. (**B**)

Technique and Reading the Test Results

98. Atopy patch tests have been evaluated in patients with atopic dermatitis and eosinophilic esophagitis as an adjunct for the diagnosis of inhalant and food allergy. (**B**)
99. Atopy patch tests for foods are prepared with dried or desiccated foods mixed into an aqueous solution and placed in 12 mm Finn Chambers before positioning on the patient's back. (**B**)
100. Atopy patch tests for the diagnosis of drug allergy are performed by incorporating liquid or powdered drugs into petrolatum or aqueous solvents, which are added to 12-mm Finn Chambers and placed on the back. (**B**)
101. Use tests have been developed for weak sensitizers (repeated open application test [ROAT]), substances with poor percutaneous absorption (strip patch test), and several premarketing dose response provocation tests for determining the minimal sensitizing dose of potential contactants in human volunteers. (**B**)

102. In the strip patch test penetration of substances is enhanced by repeated adhesive tape stripping before application of the contactant patch to the stripped area. (**B**)
103. The ROAT is an exaggerated use test designed to determine a patient's biologic threshold or response to a suspected contactant, especially if this has not been achieved with prior open or closed patch testing. (**B**)

Clinical Relevance

104. Although clinical relevance is still evolving with regard to the APT, several investigative groups have reported that this test may be an adjunct in detection of specific allergens in atopic dermatitis and eosinophilic esophagitis. (**B**)

Sensitivity, Specificity, and Positive and Negative Predictive Indices

105. The role of the atopy patch in predicting clinical allergy to food is indeterminate. (**B**)

Limitations

106. The lack of standardization of APTs for diagnosis of both food and drug allergy is the chief limitation. (**C**)

Safety

107. Although the purpose of APTs is to test for food and drug nonimmediate reactions, the possibility of anaphylaxis must be considered because there could be significant percutaneous absorption of proteins and/or simple chemicals with high anaphylactogenic potential. (**B**)

Number of Epicutaneous Skin Tests

108. The appropriate number of APTs is indeterminate because they are not routinely performed. (**B**)
109. Because ACD is frequently caused by unsuspected substances, up to 65 patch tests may be required for diagnosis. (**D**)

In Vitro Diagnostic Tests of Immediate Hypersensitivity

Total Serum IgE Assays

110. Total serum IgE concentrations are reported in international units or nanograms per milliliter (1 IU/mL = 2.44 ng/mL). (**A**)
111. Total IgE is cross-standardized with the World Health Organization (WHO) 75/502 human reference IgE serum verified by periodic proficiency surveys. (**B**)
112. The clinical applications of total serum IgE are of modest value. High serum IgE concentrations occur in allergic bronchopulmonary Aspergillosis (ABPA), the therapeutic response of which is evaluated by serial IgE values. (**B**)

113. Total serum IgE is required for assessing the suitability of a patient for omalizumab therapy and determining the initial dose. (**B**)

Allergen Specific IgE Assays

114. As with total IgE, commercial specific IgE antibody assays are calibrated using heterologous interpolation against the WHO 75/502 human IgE reference serum, thereby enabling a uniform system of reporting. (**E**)
115. In addition to WHO 75/502 calibration, an earlier specific IgE classification system was based on internal positive calibration curves from a positive control heterologous serum containing specific IgE antibodies, which in the original specific in vitro IgE (RAST) was white birch specific. However, FDA clearance for modified specific IgE tests requires use of homologous internal control allergic sera whenever this is possible to obtain. (**E**)
116. The precise sensitivity of these immunoassays compared with prick/puncture skin tests has been reported to range from less than 50% to more than 90%, with the average being approximately 70% to 75% for most studies; similar sensitivity ranges pertain when immunoassays are compared with symptoms induced after natural or controlled organ challenge tests. (**C**)
117. As with skin tests, the interpretation of specific IgE results requires correlation with the history, physical examination, and, in some cases, symptoms directly observed after natural or laboratory exposure to allergens. This cannot be accomplished by commercial remote practice laboratories, which base recommendations for immunotherapy on a history form submitted by the patient and specific IgE results. (**B**)
118. Because the constitutive allergenicity, potency, and stability are variable among commercial allergen extract reagents, sensitivity and the positive predictive value of both prick/puncture and specific IgE tests generally tend to be higher among pollens, stable anaphylactogenic foods, house dust mite, certain epidermals, and fungi compared with venoms, drugs, and chemicals. (**C**)
119. Proper interpretation of specific IgE tests needs to take into consideration variables such as the binding affinity or avidity of allergens, solid-phase systems, cross-reactive proteins and glycoepitopes, specific IgG antibodies in the test system, and high total serum IgE (>20,000 IU). (**E**)
120. A multiallergen (up to 15 allergens bound to a linear solid-phase system) test can screen for atopic status, following which allergen specific tests are required for more definitive evaluation. (**C**)
121. Specific IgE immunoassays are not recommended as a definitive confirmatory test for several specific clinical conditions. They provide neither diagnostic nor prognostic information when measured in the cord blood of newborn infants. They do not have sufficient sensitivity for foolproof prediction of anaphylactic sensitivity to venoms or penicillins. (**B**)
122. Specific IgE immunoassays may be preferable to skin testing under special clinical conditions such as widespread skin disease, patients receiving skin test suppressive therapy, uncooperative patients, or when the history suggests an unusually greater risk of anaphylaxis from skin testing. (**B**)
123. Determination of allergen specificity by inhibition of specific IgE binding is a unique attribute of specific IgE testing. (**E**)
124. Automated systems using multiplexed allergen assays are being rapidly developed. One of these is cleared by the FDA for the simultaneous measurement of 10 allergens. (**E**)

Allergen Specific IgG and IgG Subclass Assays

125. Allergen specific IgG may be measured by immunodiffusion or immunoabsorption. (**E**)
126. Immunodiffusion antibodies to cow's milk are associated with Heiner's disease, a non-IgE disorder that presents in infants with pulmonary infiltrates. (**B**)
127. IgG and IgG subclass antibody tests for food allergy do not have clinical relevance, are not validated, lack sufficient quality control, and should not be performed. (**B**)
128. Although a number of investigators have reported modest increases of IgG4 during venom immunotherapy, confirmation and validation of the predictive value of IgG4 for therapeutic efficacy of venom immunotherapy are not yet proven. (**C**)

Allergen Specific IgE Concentration

129. The probability distribution of specific IgE for several anaphylactogenic foods (peanuts, egg white, cow's milk, and codfish) can define clinical sensitivity as verified by double-blind oral challenge tests; similar relationships have been defined for several respiratory allergens. (**A**)

In Vitro Methods of Allergen Standardization

130. Although allergens can be standardized either by radioimmunodiffusion or immunoassay inhibition based on major allergenic epitopes, the FDA selected BAU instead because in vitro analytic techniques would have been variable from allergen to allergen and would have caused great confusion. (**C**)

Histamine and Leukotriene Tests

131. Histamine and leukotriene release measurements from human basophils after incubation with allergen are valuable research tools for in vitro investigations of allergy. (**B**)
132. The recent availability of several sensitive immunoassays for histamine and leukotriene C4 is a significant technological advance for measuring these mediators in various biologic fluids or release from whole blood, isolated basophils, mast cells, or other cultured cells. (**B**)
133. Histamine and its N-methyl histamine metabolite may be measured in 24-hour urine samples after suspected anaphylactic episodes. (**B**)

Plasma Tryptase

134. Plasma tryptase, particularly the beta form, should be obtained within 4 hours after an anaphylactic episode. (**B**)
135. Combined alpha and beta species of plasma tryptase are elevated in patients with systemic mastocytosis. (**A**)

Eosinophils, Eosinophil-Derived Substances, and Chemoattractants

136. Eosinophils in body fluids correlate highly with the diagnosis of allergic rhinitis, allergic asthma, and eosinophilic bronchitis. (**B**)
137. Elevated eosinophil derived substances (i.e., ECP) and chemoattractants (i.e., eotaxin) in body fluids are indicators of allergic inflammatory disease. (**B**)

Basophil Activation Test

138. A basophil activation test measured by expression of CD63 and CD203c and detected by flow cytometry is being evaluated for many IgE-mediated disorders. (**C**)

In Vitro Diagnostic Tests of Cell-Mediated Immunity

Background and Present Application

139. Tests that quantify lymphocyte function measure the ability of lymphocytes to (1) proliferate, (2) produce inflammatory mediators and cytokines or chemokines, (3) mount cytotoxic responses, and (4) regulate immune responses. (**B**)
140. Lymphocyte proliferative responses may be evaluated by either nonspecific mitogens (e.g., phytohemagglutinin, concanavalin A, or pokeweed) or specific soluble and cell-bound antigens. (**B**)
141. In vitro proliferative responses to some soluble antigens, but not mitogens, have been shown to correlate with in vivo delayed hypersensitivity. The role, however, of lymphocyte proliferation as measured in vitro in the pathogenesis of the delayed-type hypersensitivity tissue reaction is unclear. (**B**)
142. Cytokines (interleukin [IL]-1 through IL-33) and growth factors are glycoproteins produced by a variety of cells that are capable of altering activities of other cells through interaction with specific surface receptors. (**E**)
143. Chemokines are small (8 to 10 kDa) proteins secreted by many immune and nonimmune cells with essential roles in inflammatory and immune reactions, including the late-phase cutaneous response. (**E**)
144. Cytokine and chemokine profiles play essential roles in allergic inflammation and are being increasingly evaluated as phenotypic markers and in the differential diagnosis of human hypersensitivity disorders. (**B**)

Current Methods

145. Other bioactive indices of cell-mediated immunity include cytotoxic assays, cultures of mixed lymphocytes, and macrophage inhibition. (**E**)
146. Most cytokines and chemokines can be measured by commercial enzyme-linked immunosorbent assay (ELISA) and enzyme-linked immunosorbent spot (ELISpot) immunoassays. (**E**)
147. Proinflammatory cytokines or chemokines, which are particularly associated with cell-mediated immunity, include interferon-gamma, IL-12, tumor necrosis factor alpha (TNF-alpha), IL-16, macrophage inhibitory factor (MIF), macrophage inflammatory protein 1 (MIP-1), and MCP 1, 2, and 3. (**B**)

Nonspecific Screening Tests for Cellular Immune Competency

148. Simple, cost-effective tests include (1) an absolute lymphocyte count, (2) the absolute number of CD4+ T cells, and (3) the CD4+/CD8+ ratio. (**B**)

Other Diagnostic Immunologic Tests

149. Investigation of non-IgE and non–cell-mediated clinical immunologic disorders may require tests that indicate abnormal adaptive and innate immune reactions. (**B**)

Immune-Mediated Gammopathies

150. Abnormal serum and urine proteins, including cryoglobulins, may be associated with several abnormal immune syndromes. (**B**)

Nonspecific Tests of Immunologic Function

151. The inflammatory consequences induced by immune functions may be detected by nonspecific tests, such as complete blood cell count with differential, sedimentation rate, C-reactive protein, and other acute-phase reactants. In some instances, functional assays of neutrophils and macrophages may be necessary to pinpoint inflammatory responses. (**B**)

Complement Activation

152. Evaluation of complement activation with a decrease of C3 and C4 may indicate complement deficiency, drug reactions, or the presence of immune complexes, which often are associated with increases in serum cryoglobulins and C1q binding. (**B**)

Autoimmunity

153. Autoantibody profiles offer important diagnostic adjuncts in the diagnosis of collagen vascular diseases, vasculitides, and cytotoxicity disorders. (**B**)

Unproven Tests

154. Procedures for which there is no evidence of diagnostic validity include cytotoxic tests, provocation- neutralization, electrodermal testing, applied kinesiology, iridology, hair analysis, or food specific IgG, IgG4, and IgG/IgG4 antibody tests. (**B**)

Definitions:

Category of Evidence

Ia Evidence from meta-analysis of randomized controlled trials

Ib Evidence from at least 1 randomized controlled trial

IIa Evidence from at least 1 controlled study without randomization

IIb Evidence from at least 1 other type of quasi-experimental study

III Evidence from nonexperimental descriptive studies, such as comparative studies, correlation studies, and case control studies

IV Evidence from expert committee reports, the opinion or clinical experience of respected authorities, or both

LB Evidence from laboratory-based studies

Strength of Recommendations

A Directly based on category I evidence

B Directly based on category II evidence or extrapolated from category I evidence

C Directly based on category III evidence or extrapolated from category I or II evidence

D Directly based on category IV evidence or extrapolated from category I, II, or III evidence

E Directly based on category LB evidence

F Based on consensus of the Joint Task Force on Practice Parameters

NR Not rated

CLINICAL ALGORITHM(S)

None provided

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The type of supporting evidence is identified and graded for each recommendation (see "Major Recommendations").

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

- Appropriate selection and utilization of allergy diagnostic testing
- Improved quality of care by facilitation of prompt and accurate diagnosis of hypersensitivity disorders

POTENTIAL HARMS

- False-negative and false-positive test results may occur with allergy testing.
- Although adverse events occurring after intracutaneous tests are rare, they can occur. Large local reactions, both immediate and late, may cause discomfort and occasionally mild, nonprogressive systemic reactions may be associated with the latter. Immediate systemic reactions are more common with intracutaneous tests because larger volumes are injected. Six fatalities attributed to intracutaneous skin tests were reported.
- Life-threatening generalized systemic reactions are rarely caused by prick/puncture tests. In a recent retrospective survey, 1 death was reported in a patient who received 90 food prick/puncture tests at one time.
- Immediate hypersensitivity reactions, including anaphylaxis, have been reported after tuberculin skin tests.
- Repetitive (≥2) intracutaneous penicillin testing may sensitize a small number of individuals to penicillin.
- Systemic allergic contact dermatitis (ACD) after patch testing is rare, as is reactivation of patch test reactions after oral ingestion of related allergens or even by inhalation of budesonide in patients with sensitization to topical corticosteroids.
- It is possible to sensitize a patient who had not been previously sensitized to the allergen being tested. This is particularly true of plant contactants, such as poison ivy or oak and aniline dyes.
- Although the purpose of atopy patch tests (APTs) is to test for food and drug nonimmediate reactions, the possibility of anaphylaxis must be considered because there could be significant percutaneous absorption of proteins and/or simple chemicals with high anaphylactogenic potential.
- Patients receiving beta-adrenergic blocking agents and monoamine oxidase inhibitors may present special risk-benefit problems. If a systemic reaction should occur, epinephrine may not be totally effective in patients taking beta-blockers, and epinephrine may adversely affect patients taking monoamine oxidase inhibitors.

CONTRAINDICATIONS

CONTRAINDICATIONS

The concurrent use of beta-blockers and angiotensin-converting enzyme inhibitors is cited as a relative contraindication to skin testing.

QUALIFYING STATEMENTS

QUALIFYING STATEMENTS

This is a complete and comprehensive document at the current time. The medical environment is a changing environment and not all recommendation will be appropriate for all patients. Because this document incorporated the efforts of many participants, no single individual, including those who served on the Joint Task Force, is authorized to provide an official American Academy of Allergy, Asthma and Immunology (AAAAI) or American College of Allergy, Asthma and Immunology (ACAAI) interpretation of these practice parameters. Any request for

information about or an interpretation of these practice parameters by the AAAAI or ACAAI should be directed to the Executive Offices of the AAAAI, the ACAAI, and the Joint Council of Allergy, Asthma and Immunology. These parameters are not designed for use by pharmaceutical companies in drug promotion.

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Getting Better
Living with Illness
Staying Healthy

IOM DOMAIN

Effectiveness
Safety

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

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ADAPTATION

Not applicable: The guideline was not adapted from another source.

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1995 Dec (revised 2008 Mar)

GUIDELINE DEVELOPER(S)

American Academy of Allergy, Asthma and Immunology - Medical Specialty Society

American College of Allergy, Asthma and Immunology - Medical Specialty Society
Joint Council of Allergy, Asthma and Immunology - Medical Specialty Society

GUIDELINE DEVELOPER COMMENT

These parameters were developed by the Joint Task Force on Practice Parameters, representing the American Academy of Allergy, Asthma and Immunology, the American College of Allergy, Asthma and Immunology, and the Joint Council of Allergy, Asthma and Immunology.

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FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

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GUIDELINE AVAILABILITY

Electronic copies: Available in Portable Document Format (PDF) from the Joint Council of Allergy, Asthma, and Immunology (JCAAI) Web site.

Print copies: Available from JCAAI, 50 N. Brockway, Ste 3-3 Palatine, IL 60067.

AVAILABILITY OF COMPANION DOCUMENTS

None available

PATIENT RESOURCES

None available

NGC STATUS

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